CASE REPORT

EYE LESION CAUSED BY ADULT *BRUGIA MALAYI*: A FIRST CASE REPORTED IN A CHILD FROM MALAYSIA

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Abstract. We are reporting a case of an eye lesion caused by an adult *Brugia malayi*. The patient was a 3-year-old Chinese boy from Kemaman District, Terengganu, Peninsular Malaysia. He presented with a one week history of redness and palpebral swelling of his right eye. He claimed that he could see a worm in his right eye beneath the conjunctiva. He had no history of traveling overseas and the family kept dogs at home. He was referred from Kemaman Hospital to the eye clinic of Hospital Tengku Ampuan Afzan, Kuantan, Pahang, Malaysia. On examination by the ophthalmologist, he was found to have a subconjunctival worm in his right eye. Full blood count revealed eosinophilia (10%). Four worm fragments, each about 1cm long were removed from his right eye under general anesthesia. A thick blood smear stained with Giemsa was positive for microfilariae of *Brugia malayi*. A Brugia Rapid test done was positive. He was treated with diethylcarbamazine.

INTRODUCTION

Lymphatic filariasis caused by *Brugia malayi* occurs in Southwest India, China, Indonesia, Malaysia, Korea, the Philippines and Vietnam (Tsieh, 1988). Brugian filariasis is mainly a rural disease. Recent estimates of lymphatic filariasis put the global prevalence at 119 million cases (Schmidt and Roberts', 2000). *B. malayi* is responsible for 10% of lymphatic filariasis. The most prevalent form of *B. malayi* infection is nocturnal periodic, being transmitted by mosquitoes of the genera *Mansonia, Anopheles* and *Aedes*. The microfilariae (the diagnostic stage) are ingested by mosquitoes during a blood meal. They then migrate through the stomach, midgut, thoracic muscles and, finally, the mouthparts, becoming infective in about 10 days. Patients contract the disease through repeated episodes of mosquito bites. The infective larvae migrate to the lymphatic system of the host and mature in about one year, when microfilariae can be detected in the peripheral blood. In endemic areas, some people have no microfilariae and are asymptomatic, although they have the same chance of exposure to mosquito bites as do people who become infected. A considerable number of infected inhabitants have only microfilaremia and remain asymptomatic for years. The clinical manifestations in the early stage are mainly acute adenolymphangitis, with fever, headache, myalgia and pain in the arms and legs. The development of acute signs and symptoms is probably triggered by an allergic reaction to the microfilariae, unfertilized ova, molting fluid and discarded sheath. Microfilaremia and eosinophilia are usually encountered at this stage. The clinical manifestations in the chronic stage derive from obstruction of the lymphatic system as a result of a tissue reac-

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tion due to dead or dying adult worms. The salient feature is elephantiasis (lymphedema) which usually involves the limbs. Lymphatic filariasis is diagnosed by detecting microfilariae in the peripheral blood. In the nocturnal periodic form, blood should be drawn between 10:00 PM and 2:00 AM. In the sub-periodic form, microfilariae appear in the peripheral blood during the day time. In the chronic stage, when microfilariae are no longer detectable in the blood, a serologic test becomes the major tool for diagnosis (Tsieh, 1988).

The first recorded case of *B. malayi* microfilaria in a patient with uveitis was reported by Anandakannan and Gupta in 1977 in India. Rose (1966) reported an unproven case of *Brugia malayi* adult worm in the anterior chamber of the eye of a man in Malaysia. Mak *et al* (1974) reported a case of human eye infection caused by adult worms of *B. malayi* in Malaysia. Dissanaike *et al* (1974) recovered a mature female filarial worm, probably *Brugia* sp, from the conjunctiva of a man in Malaysia. In 1976, Mak and Sivanandam attempted to determine whether human ocular lesions due to *B. malayi* were due to the site of entry of the infective larvae. Cats were infected with infective larvae of *B. malayi* via ocular instillation, subconjunctival inoculation and subcutaneous inoculation. Although no conjunctival lesions were seen, infections were produced via ocular instillation, subconjunctival and subcutaneous inoculation. Adult worms were recovered from periorbital tissues and localized mainly in the lymphatic system of the head and neck regions of the cats. The results showed that the conjunctival lesions seen in humans might be due to the site of the bite of the mosquito and thus entry of the infective larvae (Mak and Sivanandam, 1976). Dissanaike *et al* in 1977 recovered an immature adult of *Dirofilaria immitis* from a human eye in Malaysia. We now report a case of subconjunctival infection of the eye caused by adult of *B. malayi*.

CASE REPORT

The patient is a 3-year-old Chinese boy from Kemaman, Terengganu, a district on the East coast of Peninsular Malaysia. He presented with a one week history of redness and palpebral swelling of his right eye. He claimed that he could see a worm in his right eye. He had no history of traveling overseas and the family kept dogs at home. From Kemaman Hospital, he was referred to the eye clinic of Hospital Tengku Ampuan Afzan, Kuantan, Pahang, Malaysia. On examination, he was clinically asymptomatic. His axillary and inguinal lymph nodes were enlarged. He was seen by an ophthalmologist, and was found to have a subconjunctival worm in his right eye. A full blood count revealed eosinophilia (10%). A peripheral blood film examination revealed microfilariae and was mistakenly diagnosed as *Dirofilaria immitis* by the staff of Veterinary Department, Kuantan, Pahang. Two days after being warded, four worm fragments, each about 1cm long were removed from his right eye under general anesthesia. No attempts were made to identify the worm. A small piece of conjunctival tissue was biopsied for histological examination and sent to the Department of Parasitology, Faculty of Medicine (FOM), University Malaya (UM). The tissue was later sent to the Department of Pathology, FOM, UM for histological examination. The histology report showed no eosinophil infiltration and no filarial parasite. Two of the blood vessels showed vasculitis with neutrophil infiltration and mild perivascular infiltration by lymphoid cells. The pathologist interpreted the findings as vasculitis. A blood specimen was also sent to the Department of Parasitology, for confirmation of the microfilariae species. A thick blood smear was done and stained with Giemsa. The microfilariae detected were diagnosed to be that of *B. malayi*. Figs 1 and 2 show the microfilariae detected in the patient's blood. The microfilaria has a sheath which stained pink with Giemsa. It has

Fig 1–Thick blood smear of patient showing microfilariae of *Brugia malayi.* Notice that the one on the right has a characteristic nucleus in the tip of its tail. Giemsa, x 400.

Fig 2–Microfilaria of *Brugia malayi.* Giemsa x 400.

overlapping nuclei with two discrete nuclei at the tail end. It does not have a smooth curve, instead it is kinky. The cephalic space is 2:1 (length:breadth). His serum tested positive with a Brugia Rapid test kit. Since there was no attempt to identify the adult worm recovered, we assumed from the findings of *B. malayi* microfilaria in the blood that the adult worm belongs to *B. malayi*. The patient was treated with diethylcarbamazine.

DISCUSSION

The three previous case reports of *B. malayi* infection from Malaysia involving the human eye occurred among adult patients and the last reported case was 32 years ago. As far as we know, this is the first record of *B. malayi* infection of the human conjunctiva in a child from Malaysia. The thick blood film taken during the day from the patient was positive for *B. malayi* microfilaria. The parasite is probably a sub-periodic strain. This is found in leaf-eating monkeys and cats where it is a zoonosis. The normal habitat of the adult *B. malayi* is the lymphatic system. The presence of the adult worm in the ectopic site, which in this patient was found in the subconjunctival region, could be due to the site of the bite of the infective mosquito around the head and neck regions.

The drug of choice for lymphatic filariasis is diethylcarbamazine (DEC). This regimen clears microfilariae from the blood and has a limited but definite effect on adult parasites. Ivermectin, a drug active in onchocerciasis, has been used in trials for therapy for lymphatic filariasis; in a single dose it appears to be as effective as DEC at clearing microfilariae (Nutman and Weller, 1998). Karam and Ottesen in 2000 reported that combined treatment using albendazole plus ivermectin or albendazole plus diethylcarbamazine has resulted in near-zero microfilaremia levels for at least one year. Based on these new developments, the World Health Assembly adopted a resolution calling on member states to work for the elimination of lymphatic filariasis as a public health problem (Karam and Ottesen, 2000).

Control is by mosquito eradication. *Mansonia* species are the major vectors in rural areas. Control of *Mansonia* mosquitoes is by destruction or removal of the aquatic vegetation.

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